Effects of Traumatic Brain Injury on Locomotor Adaptation

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Background and Purpose: Locomotor adaptation is a form of short-term learning that enables gait modifications and reduces movement errors when the environment changes. This adaptation is critical for community ambulation for example, when walking on different surfaces. While many individuals with traumatic brain injury (TBI) recover basic ambulation, less is known about recovery of more complex locomotor skills, like adaptation. The purpose of this study was to investigate how TBI affects locomotor adaptation.

Methods: Fourteen adults with TBI and 11 nondisabled comparison participants walked for 15 minutes on a split-belt treadmill with 1 belt moving at 0.7 m/s, and the other at 1.4 m/s. Subsequently, aftereffects were assessed and de-adapted during 15 minutes of tied-belt walking (both belts at 0.7 m/s).

Results: Participants with TBI showed greater asymmetry in interlimb coordination on split-belts than the comparison group. Those with TBI did not adapt back to baseline symmetry, and some individuals did not store significant aftereffects. Greater asymmetry on split-belts and smaller aftereffects were associated with greater ataxia.

Discussion: Participants with TBI were more perturbed by walking on split-belts and showed some impairment in adaptation. This suggests a reduced ability to learn a new form of coordination to compensate for environmental changes. Multiple interacting factors, including cerebellar damage and impairments in higher-level cognitive processes, may influence adaptation post-TBI.

Conclusions: Gait adaptation to novel environment demands is impaired in persons with chronic TBI and may be an important skill to target in rehabilitation.

Video Abstract Available (See Video, Supplemental Digital Content 1, http://links.lww.com/JNPT/A74) for more insights from the authors.

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INTRODUCTION

Nearly 2% of Americans have suffered a traumatic brain injury (TBI).[1] While many regain the ability to walk,[2] accumulating evidence suggests that locomotion post-TBI is suboptimal. Persons with TBI tend to walk slower, take smaller steps and strides, show greater mediolateral sway, and may step higher to clear obstacles,[3–12] than their healthy counterparts, suggesting a cautious gait strategy (although see also McFadyen et al[13]). These deficits are exacerbated when attention is divided,[4,13–17] indicating a relationship between higher cognitive processes and functional mobility. The recovery of more complex locomotor skills, like running, lags behind walking.[18,19] Thus, despite the rapid recovery of basic ambulation, other aspects of functional locomotion may be compromised.

Functional walking requires the ability to alter locomotor patterns to meet the demands of a changing environment.[20,21] Little is known about how this is affected by TBI. While some have investigated the use of anticipatory (eg, visuomotor) control to navigate obstacles,[4,9,10,12,13] less is known about whether TBI impairs the ability to compensate for systematic changes in environmental conditions that can occur in the real world, for instance, when walking on different surfaces. Adjustments to changes in the environment can take place on different time scales.[22–25] Reactive, feedback-driven changes occur almost immediately in response to a perturbation. When the perturbation is sustained, the nervous system modifies motor behavior to reduce movement errors over a longer time scale (ie, minutes to hours). This process, called adaptation, is a form of motor learning that involves feed-forward remapping of sensorimotor control, which is evidenced by behavioral aftereffects when conditions return to normal.[26,27]

Gait adaptation has been investigated using various paradigms in different neurological populations.[24,25,28–35] While the ability to make reactive changes is unaffected by damage to the cerebrum or cerebellum,[24,25,30] the ability to make longer-term motor adaptations is affected by cerebellar damage.[25,36–42] For example, Morton and Bastian[25] showed that, as a group, people with cerebellar atrophy or spinocerebellar ataxia were unable to adapt to a novel perturbation introduced by a split-belt treadmill—a treadmill with 2 belts that can drive each leg at a different speed—and they did

not show aftereffects when belts were returned to the same speed. Adaptation was more impaired in people presenting with greater ataxia, a symptom of cerebellar damage.[25] Stroke affecting the cerebral hemispheres or incomplete spinal cord injury may slow the rate of adaptation, but such damage does not prohibit the expression of aftereffects.[24,29,32–35] Adaptation rates are also slowed in neurologically intact adults when they are distracted with an additional task.[43] Interestingly, the adaptation rate of spatial coordination is affected by distraction, but temporal coordination is unaffected,[43] suggesting a role for higher cognitive processes in spatial adaptation.

Traumatic brain injury is often caused by rapid acceleration and deceleration (eg, falls, motor vehicle accidents), resulting in traumatic shearing forces that can cause extensive and multifocal damage to white matter tracts, including those in the midbrain and pons, corpus callosum, and white matter of the cerebral hemispheres.[44,45] Such diffuse damage is frequently associated with disruptions in higher cognitive processes, including the rate of information processing and attention (reviewed in the study by McAllister[46]). The purpose of this study was to examine how TBI affects the ability to adapt gait to a novel environment, which is a critical component of functional mobility. Given the apparent role of attention in locomotor adaptation,[43] we predicted that people with TBI would be slower to adapt their gait to a split-belt treadmill than would a comparison group of individuals without disabilities. We also hypothesized that adaptation of spatial coordination would be more affected by TBI than temporal coordination. Finally, we hypothesized that the ability to make reactive changes would be unaffected by brain injury. We also evaluated the relationship between symptoms of ataxia (possibly indicative of cerebellar damage) and the ability to adapt to the split-belt treadmill and store aftereffects.

METHODS

Subjects

We recruited 14 people with a history of moderate-to-severe, nonpenetrating TBI and 11 comparison participants without neurological injury or impairment. Participants with TBI were included if they were at least 6 months postinjury and able to continuously walk for at least 5 minutes unassisted. Traumatic brain injury severity was determined by Glasgow Coma Scale (GCS) scores obtained from medical records for 12 participants with TBI (Table 1). Scores can range between 3 and 15, with higher scores indicating higher levels of consciousness: severe brain injury is classified as GCS 3 to 8; moderate brain injury is GCS 9 to 12.[47] We were unable to obtain GCS scores from 2 participants with TBI. We verified a diagnosis of moderate or severe injury on the basis of other information in medical records: TBI-5 had posttraumatic amnesia lasting more than 15 days[48–50]; TBI-8 had cerebral contusions, a subarachnoid hemorrhage, and a subdural hematoma.[50] Ethical approval was obtained through the institutional review board at Einstein Medical Center and all participants gave informed consent.

Participants with TBI underwent a motor neurological examination, including the 10-m walk test to evaluate gait speed, a rating of ataxia severity using the International Cooperative Ataxia Rating Scale (ICARS),[51] and the Lower-Extremity Fugl-Meyer (FM) Assessment[52] to assess impairment. The FM was administered on the more affected leg. If this was not readily evident at the time of assessment, participants were asked if they had more difficulty with one side of their body immediately following their injury; if so, this was identified as their more affected side. If the participant did not perceive one side as being more affected, then the leg on which they had more difficulty maintaining single leg stance was designated as more affected. We also used the High Level Mobility Assessment Tool (HiMAT)[53–56] to assess high-level locomotor tasks in participants with TBI. Characteristics of the participants with TBI are shown in Table 1. Participants in the comparison group ($n = 11$; 9 male) were aged 19 to 44 years (mean $\pm$ SD $= 31.1 \pm 8.3$ years).

We note that neither the ICARS nor the FM has been validated for use in persons with TBI. The ICARS is a semiquantitative rating scale that has been validated for use in people with spinocerebellar ataxia, Friedrich's ataxia, and Multiple System Atrophy.[57,58] Although the ICARS has not been previously used in studies of persons with TBI, it has been used as a measure of ataxia in many other neurological conditions, including idiopathic pancerebellar atrophy, gluten-associated ataxia, cerebellar tumors and strokes, and multiple sclerosis.[40,59–62] The FM was developed for individuals with hemiparesis due to stroke and has been extensively used and validated with that population. Use of the FM with populations other than stroke is limited, although Platz et al[63] used FM upper extremity portion in a study of persons with multiple sclerosis, TBI, and stroke. They showed high correlation with other upper extremity motor measures and high interrater and test-retest reliability. Since we felt that it was important to characterize lower extremity motor impairment and ataxia in our participants, and since the number of motor neurological tests validated for TBI is extremely limited, we elected to use the lower extremity FM and ICARS in this study.

Experimental Design

An overview of the design is provided here, since many details of the design have previously been described elsewhere.[23–25,30,64] Participants walked on a split-belt treadmill (Woodway Waukesha, WI) with 2 belts that could be driven at the same speed ("tied-belt") or at different speeds ("split-belt"). Participants walked while holding a front hand rail, and wore a safety harness suspended from the ceiling. At the beginning of each trial, the belts were stationary and participants were not told whether the belts would be split or tied. Experiments consisted of a 5-minute "baseline" period (tied-belts: 0.7 m/s), 15 minutes of "adaptation" (split-belts: 0.7 and 1.4 m/s), and 15 minutes of "postadaptation" (tied-belts: 0.7 m/s). The less-impaired leg (as defined in the FM assessment described previously) was on the faster belt during adaptation.

Data Collection

Joint position data were collected using infrared-emitting markers (CODAmotion, Charnwood Dynamics, Rothley, UK) placed bilaterally over the fifth metatarsal head, ankle (lateral malleous), knee (lateral femoral epicondyle),

Table 1. Characteristics of Participants With TBI[a]

Sub. ID	Gender	Age (yrs)	Time Since Injury (yrs)	Glasgow Coma Scale	Preferred Gait Speed (m/s)	HiMAT	ICARS	LE Fugl-Meyer
TBI-1	M	30	5.5	3	1.6	47	5	34
TBI-2	M	32	2.5	7	1.5	46	6	33
TBI-3	F	25	3.3	6	1.4	37	8	**30**
TBI-4	M	21	1.0	4	1.6	**33**	**26**	32
TBI-5	F	35	0.5	–[b]	1.3	**27**	12	**30**
TBI-6	M	36	3.7	3	1.2	42	3	34
TBI-7	M	22	1.1	3	1.2	48	2	34
TBI-8	M	49	1.4	–[b]	1.6	45	1	34
TBI-9	F	18	3.9	3	1.2	**17**	**19**	33
TBI-10	M	31	3.0	9	1.6	46	2	34
TBI-11	F	39	5.2	7	1.5	38	1	34
TBI-12	M	18	0.9	8	1.4	46	2	34
TBI-13	M	39	4.7	3	1.7	42	**25**	**30**
TBI-14	M	21	4.6	6	1.0	46	4	33
Mean:		**29.7 ± 9.3**	**2.9 ± 1.7**	**5.2 ± 2.2**	**1.4 ± 0.2**	**40.1 ± 9.0**	**8.2 ± 8.7**	**32.8 ± 1.6**
Range:		**18 – 49**	**0.5 – 5.5**	**3-9**	**1.0 – 1.7**	**17 – 48**	**1 – 25**	**30 – 34**

Abbreviations: HiMAT, High Level Mobility Assessment Tool (out of 54; higher score = better); ICARS, International Cooperative Ataxia Rating Scale (out of 100; higher score = worse); LE Fugl-Meyer, Lower extremity Fugl-Meyer (out of 34; higher score = better).

[a]Means are given ± 1 SD. Bold scores indicate the 3 worst performers in each scale (used to define subgroups in Figure 4).

[b]No Glasgow Coma Scale score available.

hip (greater trochanter), pelvis (iliac crest), and shoulder (acromion process). The onset of stance (heel strike) and swing (toe-off) were determined by maximum and minimum limb angle excursions and confirmed with foot switches. Limb angle was calculated between a vertical axis from the greater trochanter and a vector drawn from the greater trochanter to the fifth metatarsal head: 0° limb angle indicates that the leg is positioned vertically under the body. Positive angles denote flexion and negative angles denote extension. Voltages reflecting treadmill belt speeds were recorded from treadmill motor output. Joint position and analog data (treadmill speeds and foot switches) were synchronized and sampled at 100 Hz using CODAmotion software.

Data Analysis

The leg adapted on the slow belt is referred to as the "slow leg" and the leg adapted on the fast belt leg is referred to as the "fast leg." On the basis of the previous work,[23–25,30] we first measured 2 walking parameters that were expected to change quickly using feedback-driven mechanisms: stride length and stance time. Stride length was the sagittal plane distance traveled by the ankle marker from heel strike to toe-off on one limb. Stance time was the time from heel strike to toe-off on one limb, as a percentage of total stride time on that limb.

We also assessed 3 walking parameters that were expected to "adapt," or change more gradually using predictive feed-forward mechanisms: step symmetry, center of oscillation, and phasing.[43,64–66] The calculation of these measures is described in detail elsewhere.[43,64,65] Briefly, step symmetry was based on step length: the anteroposterior distance between the malleolus markers of each leg at heel strike.[23–25,43,64,65,67] Step length was defined as slow (SL_s) or fast (SL_f) based on which leg was leading. Step symmetry calculation was as follows:

$$\text{Step symmetry} = (SL_f - SL_s)/(SL_f + SL_s)$$

Step symmetry can be altered by adapting spatial or temporal elements of coordination, or both (see prior literature for detailed explanation[43,64–66]). Center of oscillation was used to quantify spatial coordination. This was calculated stride by stride as the midpoint of the limb angle between heel strike and toe-off for each leg. Difference in center of oscillation (fast-slow) was used to evaluate symmetry (0 = symmetry). Temporal coordination was quantified as phasing: the lag time at peak cross-correlation of the limb angle trajectories over one stride cycle. Possible phasing values ranged from 0 to 1 stride cycle, with symmetric walking having a value of 0.5.

For each parameter, the change in coordination when first exposed to split-belts ("initial perturbation") was quantified within subjects as the average values from the first 3 adaptation strides. Plateau values in late adaptation were determined by averaging data from the last 30 strides. Likewise, aftereffects were averages of the first 3 postadaptation strides, and plateau postadaptation values were averages of the last 30 strides. For feed-forward parameters, adaptation and de-adaptation rates were determined by smoothing data by averaging every 3 strides, and then calculating the number of strides it took for each subject to reach the plateau value (within 1 SD).[43] More specifically, adaptation and de-adaptation rates were quantified as the first point at which 3 consecutive smoothed points (equivalent to 9 steps) fell within 1 standard deviation of the plateau value. This number was then multiplied by 3 to obtain the actual number of strides the participant took to plateau (± 1 stride).

Statistical Analysis

For all parameters, mixed-model ANOVAs were used to compare values between groups (TBI vs Comparison) at the following experimental periods: baseline, initial perturbation, adaptation plateau, aftereffects, and postadaptation plateau. Post hoc analyses on significant main effects for experiment period and period × group interactions were conducted

using a variant of the Bonferroni test called Holm's sequential Bonferroni.[68] The Levene Test for Homogeneity of Variance confirmed the assumption of equal variance. For feed-forward parameters, mixed-model ANOVAs were used to compare baseline-subtracted initial perturbation, adaptation plateau, aftereffects, and aftereffect plateau values between groups; post hoc analyses used Holm's sequential Bonferroni tests. Unpaired *t*-tests were used to compare rates of adaptation and de-adaptation between groups. For subgroup analysis of participants with TBI based on clinical test scores, we used Mann-Whitney *U* tests and applied the Holm's sequential Bonferroni correction. Statistical analyses were conducted using Matlab (Mathworks Natick, MA) or Statistica (StatSoft Tulsa, OK) and the alpha level was set at $\alpha = .05$, except during post hoc tests when it was adjustedfor multiple pairwise comparisons.

RESULTS

Subject Characteristics

Participants with TBI were largely male (10 men and 4 women) and in their 20s to 30s (mean age ± SD: 29.7 ± 9.3 years; Table 1), and the comparison group was well-matched in terms of gender (9 men and 2 women) and age (31.1 ± 8.3 years). All participants with TBI were independent walkers. Indeed, their mean preferred walking speed (1.4 ± 0.2 m/s) slightly exceeded norms previously reported for healthy adults (1.1 m/s).[69] Participants with TBI received high scores on the Lower Extremity FM Assessment. Altogether, this suggests that basic walking skills and sensorimotor function have recovered well in this sample. Nonetheless, a few participants (TBI-4, 9, and 13) received higher scores on the ICARS, indicating more severe ataxia. There was a wide range of HiMAT scores.

Reactive Feedback Control of Gait

The reactive parameters that were expected to change rapidly in a step-like function, stride length and stance time, are illustrated in Figure 1. Stride-by-stride data are shown for a representative comparison participant (A) and a participant with TBI (B). For stride length, both participants immediately lengthened strides on the fast side and shortened strides on the slow belt when belts were split; this asymmetry persisted throughout adaptation. When treadmill belts were returned to the same speed (postadaptation), stride length symmetry was immediately restored. These single-subject results are representative of group stride length difference data (Figure 1C). There was a significant main effect of experiment period, and post hoc analysis revealed that stride length difference during early and late adaption was significantly different from baseline ($P < 0.001$). Early and late postadaptations were not different from baseline. There was no main effect of group or period × group interaction.

Similar trends were observed in stance time. In adaptation, slow side stance immediately became longer than fast side stance in both participants (Figures 1A and 1B). These values did not change across the adaptation period. In postadaptation, stance time equalized. Group data for stance time difference (Figure 1C) show a significant main effect of experiment period ($P < 0.001$), where early and late adaptation values were different from baseline ($P < 0.001$), but postadaptation values were not. There was also a significant main effect of group ($P = 0.007$), but no period × group interaction. The main effect of group is likely related to the consistent negative offset of the comparison group data relative to the TBI group. The nonsignificant interaction suggests that the 2 groups did not react differently to the split-belt treadmill.

Adaptive Feed-Forward Control of Gait

Parameters that were expected to change gradually over adaptation—step length, center of oscillation, and phasing—are illustrated in Figure 2. Single-subject data from the same participants as Figure 1 are in (A) and (B). When exposed to split-belts, steps on the slow side immediately became longer than fast side steps in both participants (Figures 2A and 2B); however, step length difference early in adaptation was greater in the participant with TBI. Both participants gradually adapted step length, making values more symmetric by the end of adaptation.

Evidence of feed-forward recalibration of motor commands can be seen in both participants by the presence of aftereffects in early postadaptation: when belts returned to the same speed, the fast leg initially took longer steps than the slow leg. Group step symmetry data (Figure 2C) show a significant main effect for experiment period and a period × group interaction ($P \leq 0.005$). Post hoc tests on the interaction revealed that step symmetry in early adaptation and early postadaptation were significantly different from baseline in participants with TBI and comparison participants. In early adaptation, participants with TBI showed significantly greater step length asymmetry than comparison participants.

Adaptation of center of oscillation (Figure 2) resembles that of step symmetry. Single-subject data in (A) and (B) show that split-belts initially caused the slow leg center of oscillation to be more positive (ie, the midpoint limb angle was more flexed) and the fast leg center of oscillation to be more negative (ie, extended). There was a greater difference between fast and slow legs in the participant with TBI throughout adaptation, compared with the comparison participant. Both participants adapted back toward symmetry, and aftereffects can be seen in early postadaptation. In group center–of-oscillation difference (Figure 2C), there was a significant main effect for experiment period and a period × group interaction ($P < 0.001$). Centers of oscillation in early adaptation and early postadaptation were significantly different from baseline in both groups ($P < 0.001$). In addition, center of oscillation at late adaptation was significantly different from baseline in participants with TBI ($P < 0.001$), suggesting that this parameter had not fully adapted.

Phasing adaptation (Figure 2) was similar in the comparison participant (A) and participant with TBI (B). The phase relationship between the limbs was initially perturbed by split-belts, but both participants adapted and phasing returned to symmetric values. Aftereffects showing the opposite phase relationship were present in early postadaptation. In group data (Figure 2C), there was a main effect for experiment period ($P < 0.001$) but no main effect for group or period × group interaction. Post hoc analysis of the main effect showed significant differences between baseline and the following periods: early adaptation, early postadaptation, and late postadaptation

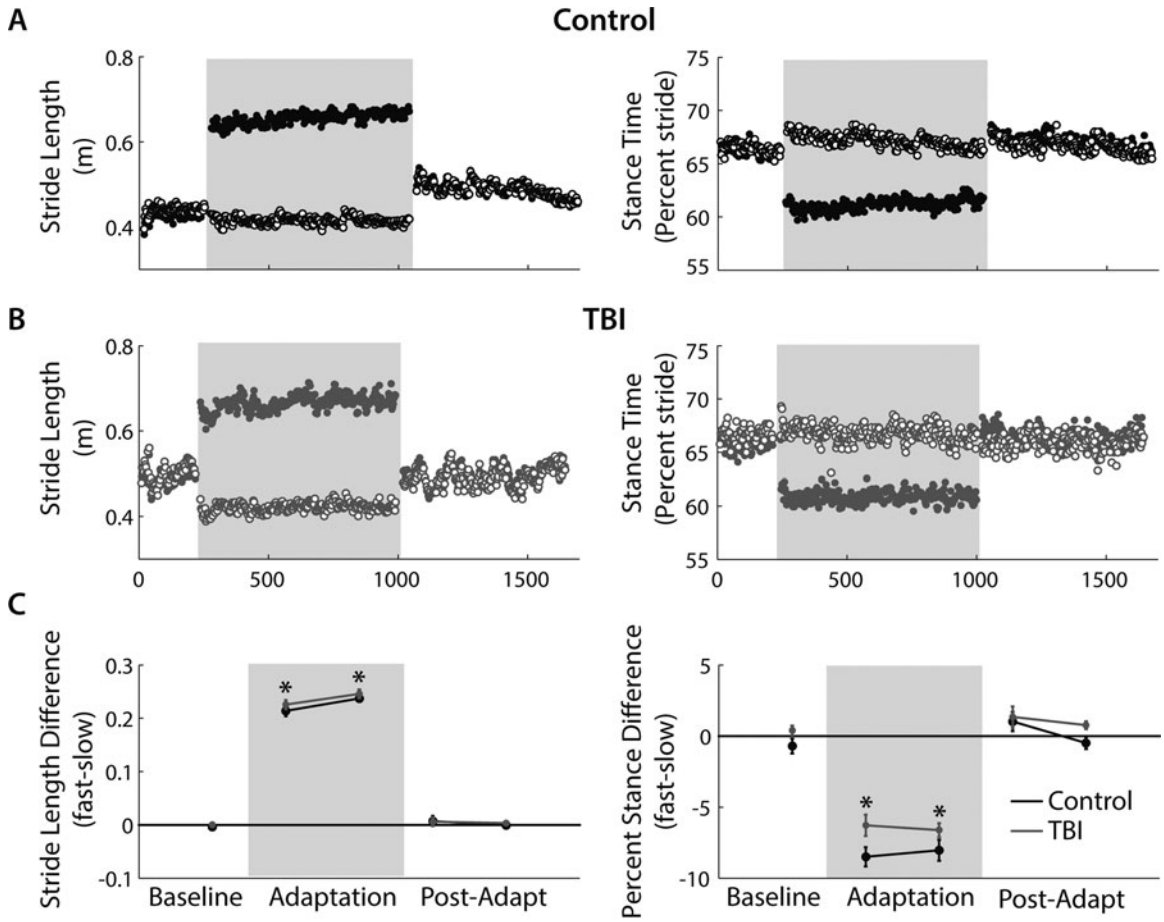

Figure 1. Reactive feedback gait modifications during split-belt walking. Experimental data from a control participant and a participant with traumatic brain injury (TBI) (Subject ID TB-14, Table 1) are shown in (A) and (B), respectively. Left-side plots show stride length and right-side plots show stance time as a percentage of total stride time (closed circles: fast leg; open circles: slow leg). Data have been smoothed by averaging every 3 strides. Initially belts are tied at 0.7 m/s (baseline). During the adaptation period (in grey box), belts are split at 0.7:1.4 m/s. This causes rapid and large changes in stride length and stance time in both subjects; these changes do not dissipate over the adaptation period. When tied-belts are reintroduced (postadaptation period; following grey box), stride length and stance time immediately return to symmetric, near-baseline values. (C) Averaged stride length difference (left) and percent stance difference (right) across all subjects. The 2 points plotted for adaptation and postadaptation are the average of the first 3 strides (early adaptation/postadaptation) and the average of the last 30 strides (late adaptation/postadaptation), respectively. There were significant main effects for experiment period in both measures, but no period × subject group interaction; asterisks indicate experiment periods that significantly differed from baseline in post hoc analysis of the main effect.

($P \leq 0.002$). It is not clear why late postadaptation values did not return to baseline symmetry; however, note that the difference between postadaptation and baseline was quite small (albeit significant).

Our analysis determined that, as a group, people with TBI were capable of adapting and storing aftereffects. However, there were also interesting differences in how participants with TBI and comparison participants adapted. To examine these differences, adaptation and postadaptation data were baseline-subtracted (within subjects) to remove any initial offsets in gait symmetry. These data were averaged across subjects (Figure 3). Similar to what was shown in Figure 2C, there was a significant difference between groups in the initial perturbation for step symmetry (Figure 3A). The initial perturbation for center of oscillation (Figure 3C) and phasing (Figure 3E) approached significance (see Figure 3 for *P* values). There were also significant differences in the adaptation plateau value for step symmetry (A) and center of oscillation (C). There were no significant differences between groups in the number of steps to reach a plateau in adaptation (red plots, Figure 3). There were no differences between groups in aftereffect size, plateau value, or the number of steps to plateau in postadaptation (Figures 3B, 3D, and 3F).

Relationship Between Clinical Assessments and Adaptive Capacities

To examine whether adaptation capacity was associated with scores achieved in the clinical assessment scales (ICARS, FM, and HiMAT), the TBI group was subdivided by scores on

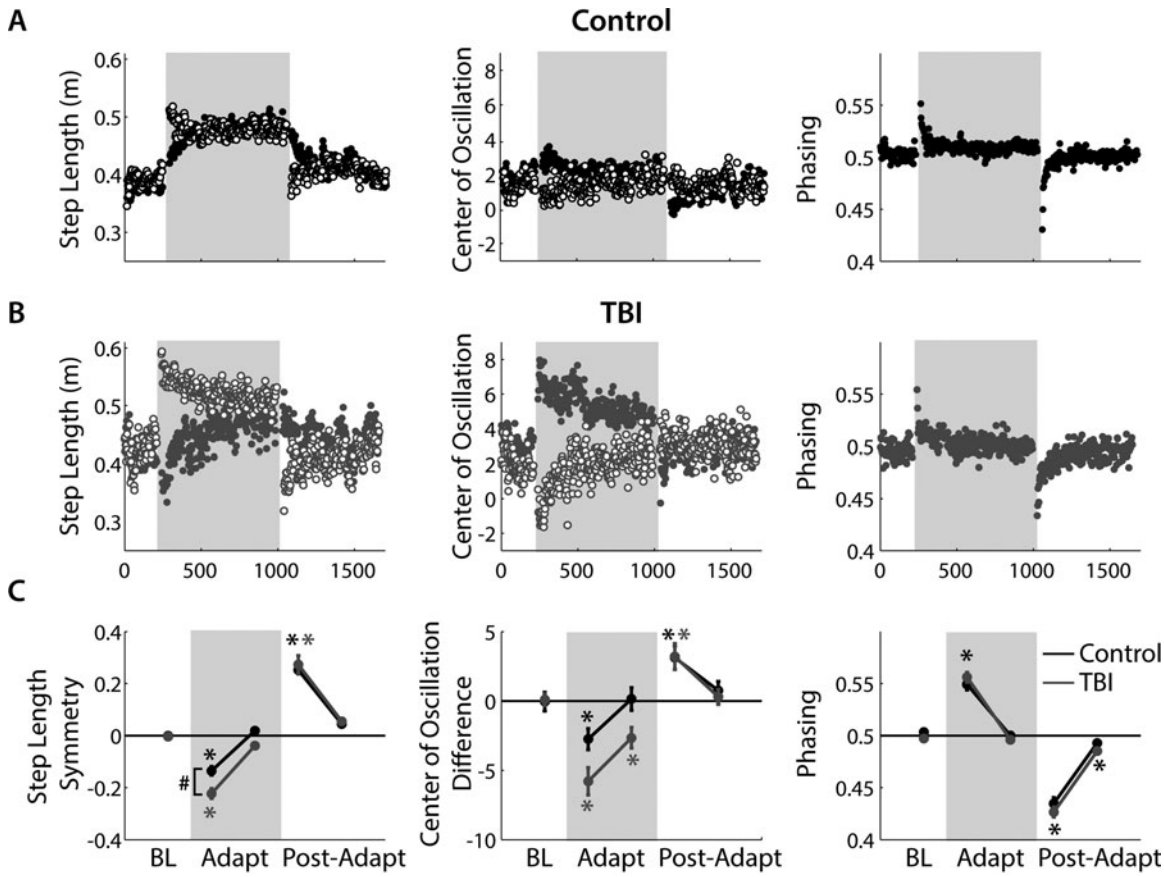

Figure 2. Adaptive feed-forward gait modifications during split-belt walking. (A) and (B) show step length (left), center of oscillation (center), and phasing (right) from the same participants as in Figure 1. Data are as plotted in Figure 1. Both the control (A) and TBI (B) participants show asymmetry in all measures when first exposed to split-belts (beginning of adaptation period, in grey box); however, the participant with TBI shows a larger asymmetry, particularly in step length and center of oscillation. Both participants gradually become more symmetric during adaptation, and they show aftereffects of comparable size in early postadaptation. (C) Averaged data across all subjects for these measures, as shown in Figure 1. There was a main effect for experiment period for all measures, and a period × group interaction for step symmetry and center-of-oscillation difference. For step symmetry and center of oscillation, asterisks mark periods that were significantly different from baseline in each group (black = control; grey = TBI). The pound sign marks a significant difference between groups. For phasing, asterisks indicate experiment periods that differed from baseline in post hoc analysis of the main effect (ie, collapsed across groups).

these tests. For each scale, the lowest-performing 3 individuals were compared with the remainder of the group. The 3 worst scores for each scale are given in bold in Table 1 for reference. Figure 4 shows how this subgrouping affects the magnitude of the initial perturbation, adaptation plateau, and aftereffect for each interlimb coordination measure. Mann-Whitney *U* tests were used to evaluate differences between groups.

Subgrouping based on ICARS scores resulted in significant differences in the initial perturbation and aftereffect size for step symmetry: people with higher ICARS scores, indicating greater ataxia, were more perturbed by split-belts and showed smaller aftereffects. A similar difference was noted in center-of-oscillation aftereffect size, although this did not reach significance. Subgrouping by FM resulted in 1 significant difference: the phasing adaptation plateau was higher in lower-scoring individuals. Subgrouping by HiMAT scores resulted in no significant differences. Although these data were baseline-subtracted, we also analyzed unsubtracted baseline data to determine whether any of the subgroups walked asymmetrically in natural conditions; no significant differences were found (data not shown).

DISCUSSION

This study is the first, to our knowledge, to examine how TBI affects locomotor adaptation, a form of motor learning that helps one reduce movement errors when the walking environment changes. Compared to the vast literature on TBI-related cognitive and behavioral impairments,[70,71] motor function remains understudied. The research that has been done, including this work, has demonstrated that dynamic balance and gait irregularities persist years after injury.[3,4,7–13,15–17] Understanding the causes and consequences of these abnormalities may help identify rehabilitation treatments to assist

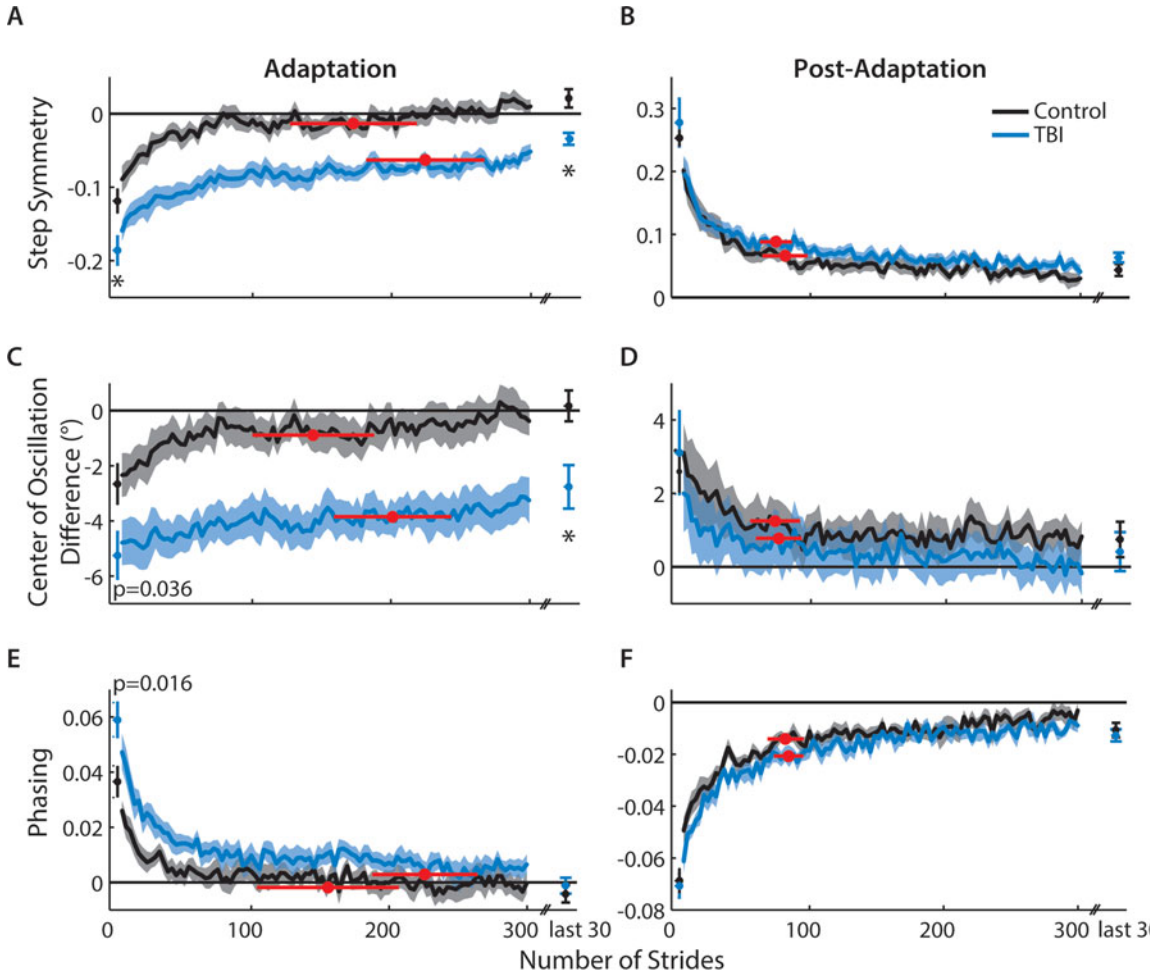

Figure 3. Averaged adaptation (left column) and postadaptation (right column) data for control (black) and traumatic brain injury (TBI) (blue) participants. (A) and (B) Step symmetry; (C) and (D) center of oscillation; and (E) and (F) phasing. For each measure, averaged baseline values have been subtracted from the data to remove any baseline offsets in symmetry; data have also been smoothed by averaging every 3 strides. The point showing averaged data from the first 3 strides is plotted separately to allow direct comparison of the initial perturbation (first 3 strides of adaptation) and aftereffect (first 3 strides of postadaptation). Averaged data from the last 30 strides (plateau value) are also plotted separately at the end of each curve. Error bars/shaded regions show standard error. The red dots show the average number of strides (± standard error) it took to reach a plateau in each curve. Asterisks mark significant differences between groups in the first 3 strides or the last 30 strides. There were no significant differences between groups in the number of strides it took to plateau.

people in recovering preinjury levels of function. This study represents a step toward this goal.

Locomotor Adaptation Post-TBI

Participants in our sample walked at speeds comparable with noninjured adults,[69] but performance in more complex locomotor function was impaired. HiMAT scores ranged from 17 to 48 out of the maximum 54, whereas the median normative HiMAT values for healthy young males and females were 54 (interquartile range: 53-54) and 51 (interquartile range: 48-53), respectively.[73] This supports previous findings that, despite well-recovered walking, people with TBI are still impaired in more challenging locomotor tasks, such as running, walking backward, and climbing stairs.[18,19]

We demonstrated that TBI did not impair the ability to make immediate feedback-driven changes in stride length or stance time on the split-belt treadmill. Prior work has shown that these parameters are also unaffected by damage to the cerebrum and cerebellum[24,25,30]; thus, our result in TBI is perhaps not surprising. Spinal reflexes that are sensitive to sensory cues, such as loading and hip position, are likely important in determining the length and timing of stance and swing phase[74,75]; if responses to sensory cues are primarily driven by spinal circuits, then one would expect these responses to remain intact following damage to supraspinal centers.

While the ability to make feedback-driven changes in stride length or stance time was preserved, some interesting and novel effects of TBI on the adaptive, feed-forward control

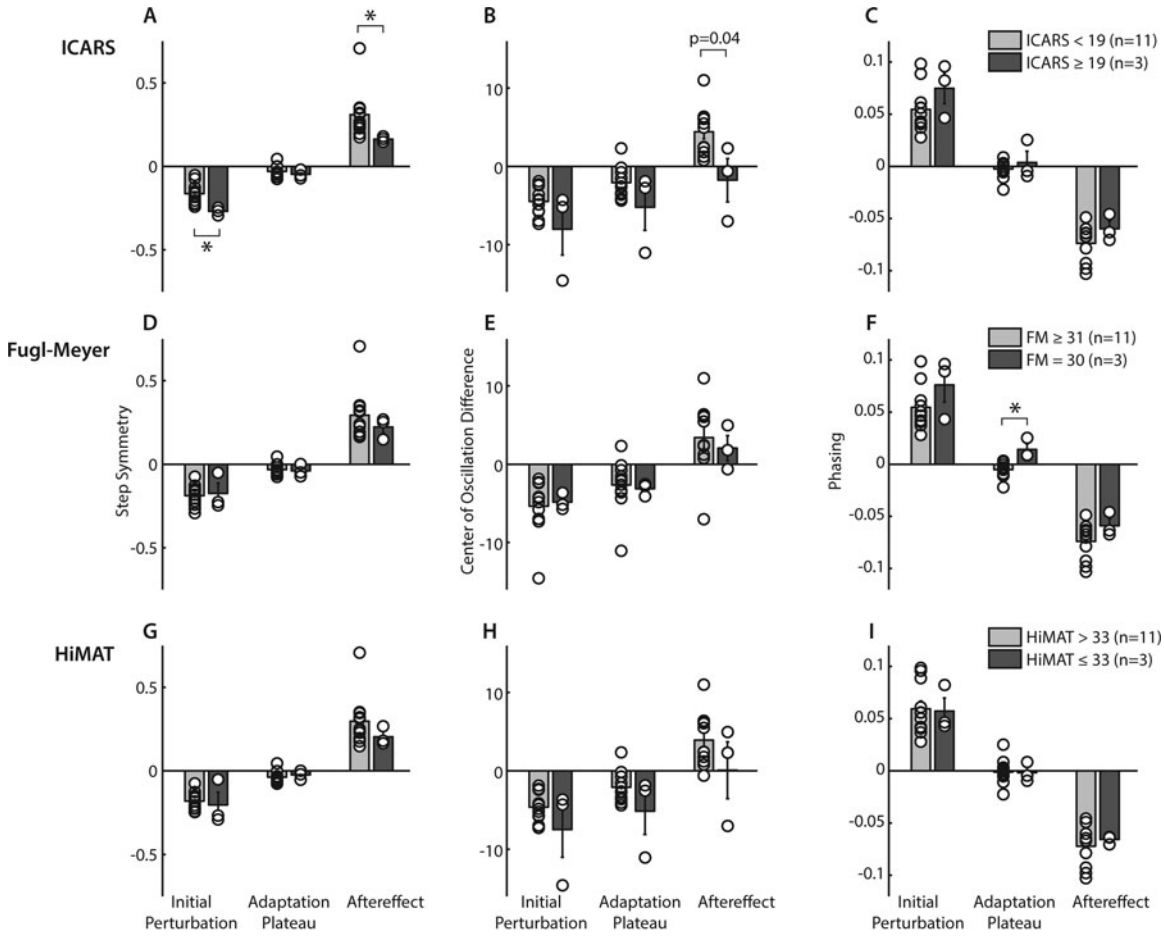

Figure 4. Relationship between clinical test scores and magnitudes of initial perturbation, adaptation plateau, and aftereffect. The 3 worst-performing participants with traumatic brain injury in each clinical scale (dark gray) were compared with the remainder of the group (light gray). (A)-(C) show grouping by International Cooperative Ataxia Rating Scale (ICARS), (D)-(F) show grouping by Fugl-Meyer, and (G)-(I) show grouping by High Level Mobility Assessment Tool (HiMAT). Columns of plots correspond to step symmetry, center of oscillation, and phasing, respectively. Bar plots show averaged group data (± standard error) and superimposed circles represent individual subject data.

of gait (ie, step symmetry, center of oscillation, and phasing) were observed. As a group, participants with TBI were able to adapt and showed significant aftereffects in spatial and temporal coordination parameters. Nevertheless, the TBI group was significantly more perturbed by split-belts, particularly in step symmetry, than the comparison groups (Figure 3). This difference in asymmetry was maintained across the adaptation period in step symmetry and center of oscillation, and participants with TBI had still not returned to baseline symmetry by late adaptation. This can also be observed in the example (TBI participant) in Figure 2B; note that step lengths and center of oscillation were not equal between the 2 sides at the end of adaptation. Phasing, in contrast, was similar between TBI and comparison groups at late adaptation.

The TBI group did not adapt back to baseline symmetry in 15 minutes of walking on a split-belt treadmill, particularly in spatial coordination. It is possible that they may have adapted back to baseline symmetry if given more time, but this question remains open to speculation. There was no significant change in step-length symmetry over the last 30 strides of the adaptation period, suggesting that there was a plateau. Future studies with longer adaptation periods would be required to conclusively test this.

Factors Influencing the Size of Initial Perturbation to Split-belts

In participants with TBI, the size of the initial perturbation on first exposure to the treadmill was large. Since this difference appears early in adaptation, it may reflect an impairment in the reactive response to split-belts. However, since we saw no difference in stride length and stance time asymmetry between TBI and comparison groups, we believe that this is unlikely. Alternatively, a deficit in adaptive capacity may underlie this difference. Motor adaptation has an initial fast-learning phase and a longer-term slow-learning phase.[72] It is possible that TBI impairs the fast-learning phase, during which motor commands are rapidly recalibrated to compensate for large movement errors.[72] In our study, there were 2 to

4 strides that were not analyzed at the beginning of each trial as the treadmill belts accelerated to the desired speed. During this time, participants received information about the speed of the belts. If participants in the comparison group were able to recalibrate motor commands faster than participants with TBI in these first few strides, then this may explain the initial difference in early adaptation. A study of locomotor adaptation in people with poststroke hemiparesis also showed that their rate of adaptation was slowed compared with participants in the comparison groups; however, in contrast to this study, the differences were noted in the later, slow-learning phase.[29] The earlier phase of locomotor adaptation can be slowed in healthy participants by distracting them with a secondary task.[43] The fast-learning phase is thought to be under more conscious or executive control than later phases,[76] which could also explain why differences between participants in the comparison group and participants with TBI emerge early in adaptation. Thus, it is possible that the adaptation rate is affected by attention, which is known to be impaired in persons with TBI.[46]

Little is known about how higher-level cognitive processes, including attention, interact with locomotor function in people with TBI. In one study, Cantin et al[5] reported a significant relationship between scores in the Trail Making B test and the height of foot clearance over an obstacle in people with TBI: those who took longer to complete the Trail Making B test tended to step higher over obstacles. The Trail Making B test is an assessment of visual attention and task switching and may correlate with the ability to perform visuospatial navigation tasks.[5] It is not clear whether a similar relationship between trail making and split-belt treadmill adaptation would be expected, since this form of locomotor learning is not reliant upon vision.[77] Given the prior finding that attention affects split-belt adaptation rates in healthy participants,[43] it is possible that higher-level cognitive processes can affect adaptation rates in people with TBI.

Factors Influencing the Storage of Aftereffects

Although they did not adapt back to baseline symmetry, most participants with TBI still showed evidence of adaptation; there was a sensorimotor remapping to reduce motor errors (ie, asymmetry) on the split-belt treadmill. This remapping was stored and remembered when participants returned to tied-belts, resulting in aftereffects. We found that participants in our sample who had greater symptoms of ataxia had smaller aftereffects in step symmetry. They also showed greater initial perturbations than participants with lower ataxia, perhaps reflecting impaired ability to rapidly adapt to split-belts in the fast-learning phase. Morton and Bastian[25] showed that people with cerebellar damage and high ratings of ataxia did not adapt or store aftereffects. Although we do not have direct evidence of cerebellar damage in our participants (ie, imaging), radiological and animal studies have shown that the cerebellum is often indirectly affected by TBI.[78–80] Cerebellar symptoms such as ataxia can emerge in TBI survivors with lesions in the brainstem or thalamus, perhaps as a consequence of damage to cerebellar pathways.[81,82] Atrophy and metabolic changes in the cerebellum can also occur postinjury as a consequence of diffuse axonal injury.[83,84] Although we identified some significant relationships between ataxia and adaptation, it is worth pointing out that there was also a significant difference in phasing adaptation plateau based on FM scores, which we are unable to explain. It is possible that this is a random effect of a small sample size. While not conclusive, these results suggest a potential relationship between ataxia symptoms and the ability to adapt.

Limitations

For this study, we restricted participation to participants with TBI who were able to walk for at least 5 minutes. We obtained a sample of individuals with TBI who had recovered basic ambulatory and lower limb motor function quite well (for instance, see gait speed in Table 1). It is not unusual for people with TBI to recover walking: one study reported that 73% of people who sustained a severe injury were community ambulators within 5 months.[2] While others have reported slower gait speeds post-TBI,[5–7,9,11,12] this was not true for our sample. For this reason, it is uncertain whether the results of this study can be extended to the larger TBI population, particularly to those who are less mobile. Nonetheless, we believe that there are some findings that can be extended to the high-functioning TBI population, including the observation that the recovery of normal walking speed over level ground does not preclude the possibility of other locomotor impairments.

It is also acknowledged that TBI is a heterogeneous disorder, in which both focal and diffuse injuries are brought about by a variety of pathophysiologic processes (reviewed elsewhere[85,86]) and are not always evident on clinical CT and MRI scans.[87] Because of the heterogeneity of TBI and the limited correlation of clinical presentation with imaging findings, we have used the ICARS, lower-extremity FM, and HiMAT to characterize the impairment and motor performance of our participants. While these tests provide valuable information about the severity of different motor impairments, the fact that the ICARS and FM have not been validated in this population is recognized as a limitation. Validating these scales or creating alternative scales to characterize motor impairments in this population would be important work for the future.

A final limitation is the lack of specific measures of cognitive function, including attention for participants with TBI. This study was not designed to elucidate these potentially complex relationships, and this would be a productive area for future research.

CONCLUSIONS

The gait of participants with TBI was made more asymmetric by split-belt walking than that of participants in the comparison group. This suggests a diminished ability to rapidly modify locomotor coordination in response to environmental changes following TBI. Multiple interacting factors, including cerebellar damage and impairments in higher-level cognitive processes, may influence adaptation post-TBI; this study represents a first examination of a few of these factors. This study demonstrates that adaptation to novel environmental demands is impaired in TBI survivors who are considered community ambulators, indicating that while walking speed may have recovered, other components of functional mobility have not.

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